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CASE REPORT
Idiopathic Spinal Cord Herniation: Case Report and Review of the Literature

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Abstract

Background: Idiopathic spinal cord herniation (ISCH) is a rare cause of progressive myelopathy frequently present in Brown-Séquard syndrome. Preoperative diagnosis can be made with magnetic resonance imaging (MRI). Many surgical techniques have been applied by various authors and are usually reversible by surgical treatment.

Methods: Case report and review of the literature.

Findings: A 45-year-old woman with Brown-Séquard syndrome underwent thoracic MRI, which revealed transdural spinal cord herniation at T8 vertebral body level. During surgery the spinal cord was reduced and the ventral dural defect was restorated primarily and reinforced with a thin layer of subdermal fat. The dural defect was then closed with interrupted stitches.

Results: Although neurologic status improved postoperatively, postsurgical MRI demonstrated swelling and abnormal T2-signal intensity in the reduced spinal cord. Review of the English language literature revealed 100 ISCH cases.

Conclusions: ISCH is a rare clinical entity that should be considered in differential diagnosis of Brown-Séquard syndrome, especially among women in their fifth decade of life. Outcome for patients who initially had Brown-Séquard syndrome was significantly better than for patients who presented with spastic paralysis. Although progression of neurologic deficits can be very slow, reduction of the spinal cord and repair of the defect are crucial in stopping or reversing the deterioration.

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Key Words: Brown-Séquard syndrome; Myelopathy; Spinal cord herniation, Dural defect; Spasticity; Duraplasty; Pseudomeningocele

INTRODUCTION

Idiopathic spinal cord herniation (ISCH) is a rare cause of progressive myelopathy. Brown-Séquard syndrome can be the initial presentation of ISCH (1–4). From the first published case by Wortzman in 1974, 100 cases have been reported by various authors in the English literature (1–51) (Table 1). The hypotheses regarding the pathophysiology, treatment strategies, and postoperative courses are discussed in these reports. The popularization of magnetic resonance imaging (MRI) and an awareness of the clinical setting helped achieve an accurate diagnosis of ISCH. Thus, the frequency of reports of ISCH in the literature has increased since 2000. The pathogenesis of thoracic spinal cord herniation has been ascribed to a dural defect, either congenital or acquired, in the anterior surface of the dural sac (5).

However, the exact cause of the dural defect is still not known. Different surgical techniques have been recommended to treat this disease. In our review, we identified 3 main surgical treatment strategies: (a) use of primary sutures to close the dural defect (6–9), (b) use of a dural graft to close the defect (3,10–17), and (c) enlargement of the dural defect (1,18–21).

We report an ISCH case treated with primary sutures with a good clinical outcome. We also review, summarize, and discuss the clinical findings, surgical procedures, and surgical outcome for reported cases in the literature.

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CASE REPORT

History

A 48 year-old woman presented with a 2-year history of burning sensation in the right lower limb, bilateral hip and leg pain, and gait disturbance. The patient was

Table 1. Idiopathic Spinal Cord Herniation Cases Reported in the English Literature from 1974 to 2006

	Authors	Age (y)	Gender	Symptoms/Signs	Level	Type of Intervention	Outcome (Follow-up Period)	Case No.
1	Wortzman, 1974 (27)	63	M	Brown-Séquard	T7	Primary suture	Improved (2 y)	1
2	Masuzawa, 1981 (29)	36	F	Brown-Séquard	T4-T5	Duraplasty	Improved (3 y)	1
3	Oe, 1990 (22)	61	M	Spastic paralysis	T4-T5	Resection of inner dura	Unchanged (unknown)	1
4	Isu, 1991 (24)	43	F	Brown-Séquard	T5-T6	Resection of cyst	Improved (unknown)	2
5		45	F	Spastic paralysis	T2-T3	Resection of cyst	Improved (unknown)	
6	Tronnier, 1991 (10)	45	F	Numbness	T3-T4	Duraplasty	Improved (4 mo)	1
7	Nakazawa, 1993 (23)	43	F	Brown-Séquard	T2	Resection of inner dura	Improved (4 y)	2
8		39	F	Brown-Séquard	T4	Resection of inner dura	Improved (unknown)	
9	White, 1994 (11)	61	F	Brown-Séquard	T4	Duraplasty	Unchanged (unknown)	2
10		39	M	Brown-Séquard	T8	Duraplasty	Unchanged (unknown)	
11		68	F	Brown-Séquard	T7-T8	Primary suture	Improved (1y)	
12	Borges, 1995 (7)	68	M	Brown-Séquard	T2-T3	Primary suture	Improved (7 d)	3
13		48	F	Brown-Séquard	T7	Primary suture	Improved (2 mo)	
14	Kumar, 1995 (12)	38	M	Brown-Séquard	T7-T8	Duraplasty	Improved (2 mo)	1
15		57	F	Brown-Séquard	T6	Primary suture	Improved (unknown)	
16	Hausmann, 1996 (30)	36	M	Spastic paralysis	T6-T7	Resection of hernia	Deteriorated (unknown)	4
17		51	F	Brown-Séquard	T6-T7	Not performed	No details available	
18		49	M	Spastic paralysis	T4-T5	Not performed	No details available	
19	Matsumura, 1996 (38)	63	F	Brown-Séquard	T3-T4	Resection of inner dura	No details available	1
20	Miura, 1996 (6)	49	M	Spastic paralysis	T5-T6	Resection of inner dura	Improved (6 mo)	1
21	Sioutos, 1996 (13)	34	F	Spastic paralysis	T6-T7	Duraplasty	Improved (6 mo)	1
22	Slavotinek, 1996 (31)	22	F	Brown-Séquard	T5	Reduction only, second operation	Improved (unknown)	1
23	Baur, 1997 (39)	66	F	Brown-Séquard	T10	Suture inner dura	No details available	1
24	Henry, 1997 (14)	30	F	Brown-Séquard	T7	Duraplasty	Improved (5 mo)	1
25	Uchino, 1997 (8)	71	F	Brown-Séquard	T4-T5	Primary suture	Unchanged (unknown)	2
26		61	F	Brown-Séquard	T6	Incomplete	Unchanged (unknown)	
27	Dix, 1998 (5)	44	F	Brown-Séquard	T7-T8	Duraplasty	Improved (6 mo)	1
28	Miyake, 1998 (25)	45	F	Brown-Séquard	T3-T4	Duraplasty	Improved (1 mo)	2
29		53	M	Brown-Séquard	T2-T3	Duraplasty	Improved (1 mo)	
30	Watters, 1998 (9)	55	F	Brown-Séquard	T3-T4	Closure	Deteriorated (4 mo)	1
31	Brugieres, 1999 (16)	54	F	Brown-Séquard	T6	Duraplasty	Improved (unknown)	2
32		70	M	Brown-Séquard	T5-T6	Resection of inner dura	Improved (unknown)	
33	Marshman, 1999 (26)	55	F	Brown-Séquard	T8	Duraplasty	Improved (1 y)	1
34		28	F	Brown-Séquard	T3-T4	Reduction	Improved (2 mo)	
35	Vallee, 1999 (32)	58	F	Brown-Séquard	T4-T5	Reduction	Improved (2 mo)	4
36		40	F	Brown-Séquard	T5-T6	Duraplasty	Unchanged (6 mo)	
37		49	F	Brown-Séquard	T4-T5	Duraplasty	Unchanged (6 mo)	
38	Verny, 1999 (40)	28	F	Brown-Séquard	T3-T4	Reduction, duplication	Improved (2 mo)	2
39		58	F	Brown-Séquard	T4-T5	Reduction, duplication	Improved (2 mo)	
40	Bartolomei, 2000 (41)	61	F	Brown-Séquard	T3-T4	Duraplasty	Improved (3 mo)	1
41	Ewald, 2000 (34)	51	F	Brown-Séquard	T5-T6	Duraplasty	Unchanged (2 mo)	1
42	Martin, 2000 (42)	31	M	Brown-Séquard	T8	Posterior arachnoid cyst resection	Improved (unknown)	1

Table 1. Continued

	Authors	Age (y)	Gender	Symptoms/Signs	Level	Type of Intervention	Outcome (Follow-up Period)	Case No.
43	Tekkok, 2000 (33)	49	F	Brown-Séquard	T3-T4	Duraplasty	Unchanged (5 mo)	1
44		59	M	Brown-Séquard	T4-T5	Resection of inner dura	Improved (4 y)	
45	Wada, 2000 (4)	48	M	Brown-Séquard	T5-T6	Resection of inner dura	Improved (4 y)	3
46		63	F	Brown-Séquard	T3-T4	Resection of inner dura	Improved (4 y)	
47	Kawachi, 2001 (15)	53	M	Brown-Séquard	T10	Reduction	Improved (unknown)	1
48	Miyaguchi, 2001 (1)	54	F	Brown-Séquard	T3-T4	Duraplasty	Improved (6 mo)	1
49	Morokoff, 2001 (43)	33	F	Brown-Séquard	T8	Incomplete	Improved (3 mo)	1
50	Pereira, 2001 (44)	55	M	Spastic paralysis	T2-T3	Diverticulum filled by fibrin glue	Improved (unknown)	1
51		43	F	Brown-Séquard	T4	Enlarged defect	Improved (13 y)	
52		39	F	Brown-Séquard	T3	Enlarged defect	Improved (11 y)	
53		54	F	Brown-Séquard	T4	Enlarged defect	Improved (2 y)	
54		71	F	Paraparesis	T4	Enlarged defect	Deteriorated (2 y)	
55	Watanabe, 2002 (19)	49	M	Brown-Séquard	T4	Enlarged defect	Improved (1,8 y)	9
56		47	F	Brown-Séquard	T5-T6	Enlarged defect	Improved (1,5 y)	
57		78	F	Paraparesis	T4	Enlarged defect	Improved (1 y)	
58		56	M	Brown-Séquard	T6	Enlarged defect	Improved (8 mo)	
59		47	F	Paraparesis	T3	Enlarged defect	Improved (5 mo)	
60		44	M	Numbness	T8-T9	Enlarged defect	Improved (1 y)	
61	Aizawa, 2001 (18)	60	F	Brown-Séquard	T4-T5	Enlarged defect	Improved (1 y)	3
62		59	F	Brown-Séquard?	T4-T5	Enlarged defect	Improved (1 y)	
63	Barbagallo, 2002 (28)	28	F	Spastic paralysis	T6	Closure by prosthetic material	Unchanged (6 mo)	2
64		64	M	Paresthesia	T6	Closure by prosthetic material	Deteriorated (12 h)	
65	Gellerini, 2002 (35)	53	M	Brown-Séquard	T8-T9	Duraplasty (patch)	Improved (unknown)	2
66		57	F	Brown-Séquard	T4-T5	Duraplasty (patch)	Improved (unknown)	
67		63	M	Brown-Séquard	T5–6	Conservative	Unchanged (8 y)	
68		39	F	Brown-Séquard	T6-T7	Duraplasty	Improved (3 y)	
69		50	M	Leg numbness	T4	Conservative over 6 y	Unchanged (6 y)	
70	Massicotte, 2002 (2)	44	F	Spastic paralysis	T6	Duraplasty	Deteriorated (3 y)	8
71		33	F	Numbness	T7-T8	Conservative over 6 y	Unchanged (6 y)	
72		57	F	Spastic paralysis	T6	Duraplasty 1 y	Unchanged (1 y)	
73		27	M	Spastic paralysis	T9	Duraplasty	Improved (7 mo)	
74		46	F	Numbness, pain	T4-T5	Conservative over 1 mo	Unchanged (1 mo)	
75	Sagiuchi, 2003 (45)	48	M	Brown-Séquard	T7-T8	Releasing cord	Improved (unknown)	1
76	Nakagawa, 2003 (46)	77	F	Brown-Séquard	T6-T7	Membrane suraplasty polytetrafluoethylene	Deteriorated « improved (1 y)	1
77	Sasaoka, 2003 (20)	57	M	Leg pain	T2-T3	Defect	Improved (unknown)	1
78		61	M	Brown-Séquard	T7	Duraplasty (neuropatch)	Improved (1 y)	
79	White, 2004 (3)	62	F	Brown-Séquard	T6-T7	Duraplasty (graft)	Unchanged (1 y)	3
80		66	F	Brown-Séquard	T7	Duraplasty (graft)	Improved (9 mo)	
81	Maruichi, 2004 (47)	53	F	Numbness and pain	T4-T5	Diverticulum-membrane wrapped	Improved (unknown)	1
82	Najgar, 2004 (36)	32	M	Brown-Séquard	T8-T9	Widening dural defect/duraplasty (polytetrafluoethylene)	Deteriorated « improved (8 y)	1

Table 1. Continued

	Authors	Age (y)	Gender	Symptoms/Signs	Level	Type of Intervention	Outcome (Follow-up Period)	Case No.
83	Saito, 2004 (48)	68	F	Monoparesis	T6-T7	Defect/resection of inner layer	Improved (unknown)	1
84	Aquilina, 2004 (21)	37	F	Brown-Séquard	T4	Defect	Improved (unknown)	1
85	Sugimoto, 2005 (49)	48	M	Spastic paralysis?	T4-T5	Incision of inner dura	Improved (unknown)	1
86	Karadeniz-Bilgili, 2005 (50)	36	F	Brown-Séquard	Unknown	Unknown	Improved (unknown)	1
87		31	F	Progressive paraparesis	T8-T9	Reduction cord intradurally, no repaired dura	Improved (24 mo)	
88		54	F	Brown-Séquard	T4-T5	Duraplasty (patch)	Improved (3 wk)	
89	Maira, 2006 (17)	45	F	Brown-Séquard	T4-T5	Duraplasty (patch)	Deteriorated « Improved (4 y)	5
90		50	M	Slowly progressive paraparesis	T6-T7	Reduction cord intradurally, no repaired dura	Improved (unknown)	
91		57	F	Brown-Séquard	T4-T5	Duraplasty (patch)	Improved (1 y)	
92	Saito, 2006 (51)	57	M	Brown-Séquard	T2-T3	Duraplasty (graft)	Improved (unknown)	1
93		65	F	Brown-Séquard, urinary dysfunction	T4-T5	Dural patch	Unchanged (60 mo)	
94		32	M	Bowel urinary dysfunction (ASIA-C)	T7-T8	Dural patch	Incomplete improved (147 mo)	
95		54	F	Brown-Séquard, urinary bowel dysfunction	T2-T3	Dural patch	Incomplete improved (16 mo)	
96	Barrenechea, 2006 (37)	60	F	Brown-Séquard	T2-T3	Dural patch	Incomplete improved (42 mo)	7
97		59	F	Brown-Séquard, urinary dysfunction	T5-T6	Dural patch	Incomplete improved (40 mo)	
98		34	M	Paresis (ASIA-D)	T7-T8	Dural patch	No change (30 mo)	
99		72	M	Brown-Séquard	T4-T5	Dural patch	No change (10 mo)	
100	Sasani, 2006 (present study)	48	F	Brown-Séquard	T8	Primary suture	Improved (6 mo)	1

From references 1–31; 33–52.

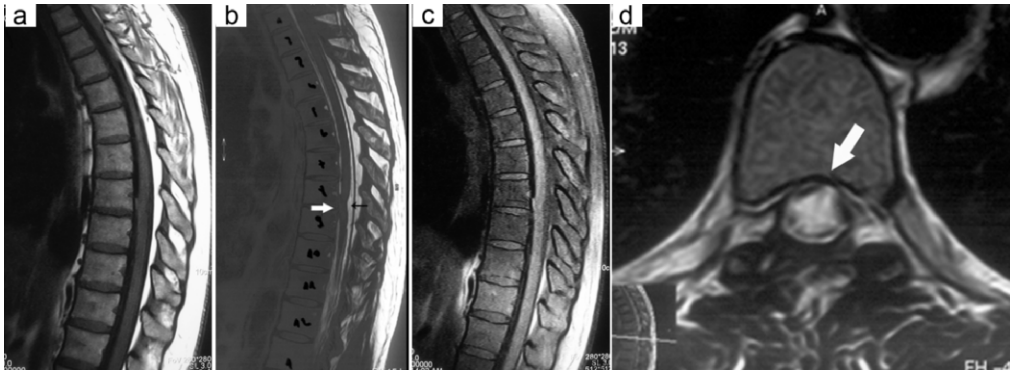

Figure 1. *Preoperative MRI. Sagittal (a) T1-weighted, (b) T2-weighted, and (c) fat-saturated T1-weighted images show ventral displacement of the spinal cord and enlarged dorsal subarachnoid space at T7-T8 (narrow arrow). (d) Axial T2-weighted image showing leftward anterior displacement of the spinal cord.*

unable to walk short distances without a cane. Orthopedic examination and radiographs were unremarkable, and she was referred for physical therapy. Her complaints persisted, and 6 months prior to presentation she experienced leg weakness and more severe hip pain on the left. Meanwhile, she also noticed loss of perception of temperature sensation in her right leg, especially while in the shower. Bladder function was normal, and she reported no history of spinal surgery or trauma.

Examination

Neurological examination revealed Brown-Séquard syndrome below the level of T8. She had diminished tactile and pinprick sensation and reduced temperature sensation in the right lower extremity. Position and vibration senses were preserved bilaterally, and reflexes were all brisk. She had leg muscle weakness in the left leg, without any muscle bulk loss. Plain radiographs of the thoracic spine were unremarkable. MRI of the thoracic spine revealed leftward anterior displacement of the spinal cord. Slight hyperintense signal was noted in the thinned spinal cord parenchyma on T2-weighted images (Figure 1). Phase-contrast MRI showed a normal subarachnoid flow pattern and confirmed the absence of a posterior arachnoid cyst in the enlarged dorsal subarachnoid space. ISCH was diagnosed on the basis of clinical and radiological findings.

Surgical Findings

Spinal surgery was performed under microscopic visualization. After a laminectomy at T8, the dorsal dura was first opened and the dentate ligament was incised on both sides. The spinal cord was displaced to the anterior left side and was adhered to the ventral dura. The operating table was then adjusted to a left 45 degrees–left oblique position. Adhesion of the spinal cord was released by blunt dissection from the anterior dural defect, and the spinal cord was carefully reduced intradurally. The 5 $\times$ 17-mm ventral dural defect was observed. The anterior epidural space was reinforced by a thin layer of subdermal fat that was affixed to the posterior longitudinal ligament with 2 stay sutures. The ventral dural defect and dorsal dura were closed with interrupted stitches.

Postoperative Course

The patient's left hip pain and bilateral leg weakness were relieved postoperatively. One week after the operation, she was able to walk without a cane with the help of the physical treatment team, and gradual improvement was noted in the weeks after the surgery. She noticed progressive improvement in thermal sensibility, and after 3 months, heat and cold tests revealed complete recovery. Three months later, postoperative MRI revealed normal position of the spinal cord and the presence of slight swelling and T2-hyperintense parenchymal signal at the level of previous herniation (Figure 2). Thirty months postoperative, follow-up MRI demonstrated no changing in reactive gliosis signal (Figure 3).

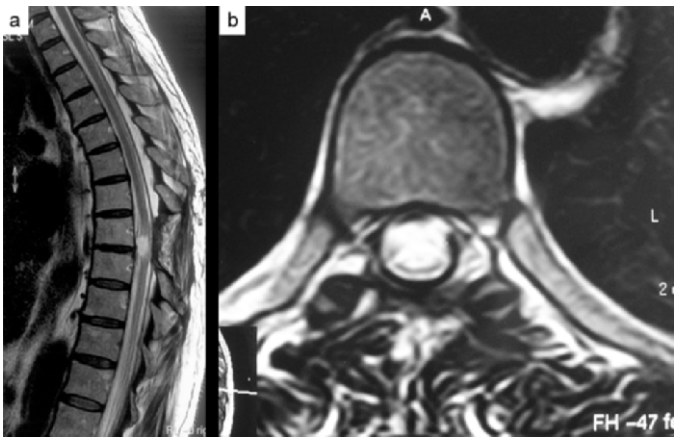

Figure 2. *(a) Three-month postoperative dorsal MRI showing the spinal cord in an anatomical intradural location with reactive gliosis signal. (b) Axial MRI showing hyperintense parenchymal signal with reduced cord expansion.*

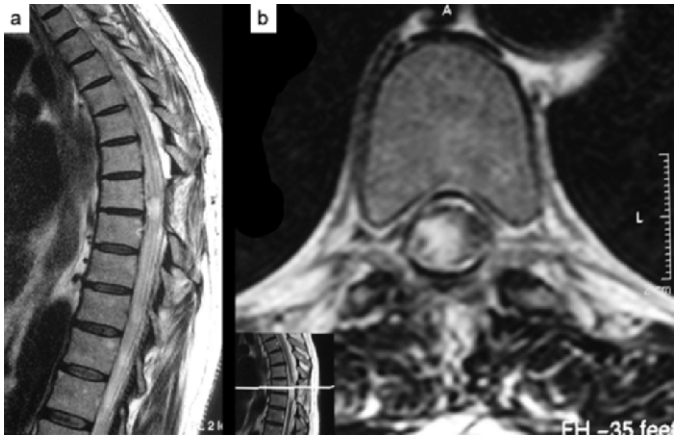

Figure 3. *Thirty-month postoperative (a) sagittal and (b) axial dorsal MRI T2-weighted scans demonstrate reactive gliosis signal due to preoperative period squeezing of spinal cord by the dural defect.*

DISCUSSION

ISCH is a spontaneous displacement of thoracic cord through an anterior dural defect. Although several theories have been proposed to explain the cause of this rare condition, the pathogenesis remains unclear. Aizawa (18) described 3 types of ventral dural defect: (a) a defect in the inner layer of the duplicated ventral dura; herniated spinal cord is covered by an outer layer of dura (16,22,23); (b) direct epidural spinal cord herniation through a full-thickness dural defect (7,10); and (c) an epidural cyst or pseudomeningocele (8,24–26). In this patient, the dural defect was full thickness and the spinal cord was herniated directly into the epidural space.

ISCH was first reported by Wortzman et al (27) in 1974 and subsequently in small series from different authors. The number of published cases in the English language literature markedly increased after 2000; there are now 100 reported cases (Figure 4). Awareness of the clinical setting and the wider use of MRI in myelopathy are considered the pertinent factors in this increase. In the literature, clinical findings of ISCH are nonspecific but characteristic, and the radiologic diagnosis is both characteristic and specific when adequate examination was performed (5,6,13).

Most spinal cord herniations occur in the thoracic spine. Involvement of the thoracic spine can be explained by normal kyphosis of thoracic spine, natural anterior position of thoracic spinal cord, the spinal cord's physiological anterior movement secondary to cardiac and pulmonary actions, and the impact of flexion and extension movements. Barbagallo et al (28) stated that outcome was less favorable for spinal cord herniation at a vertebral body level than for disk-level herniation. Review of the literature revealed that T4-T5 is the most frequently involved disk level (19%) (Figure 5). Mean age at presentation was 53 years (range 22–78 years). Female predominance was apparent, with a 67/33 female/male ratio.

In ISCH, the spinal cord is frequently shifted ventrolaterally and sometimes rotated toward the side of tethering. Tethering of the spinal cord at the side of the herniation results in unilateral damage of the lateral

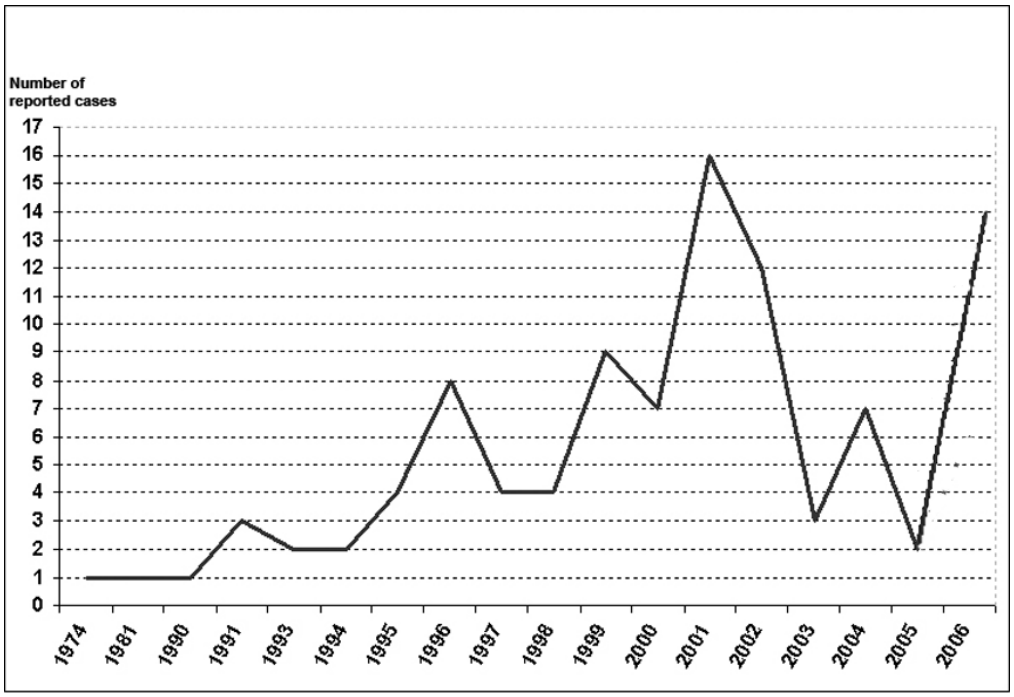

Figure 4. *Distribution of reported idiopathic anterior thoracic spinal cord herniation according to disk level and vertebral body level.*

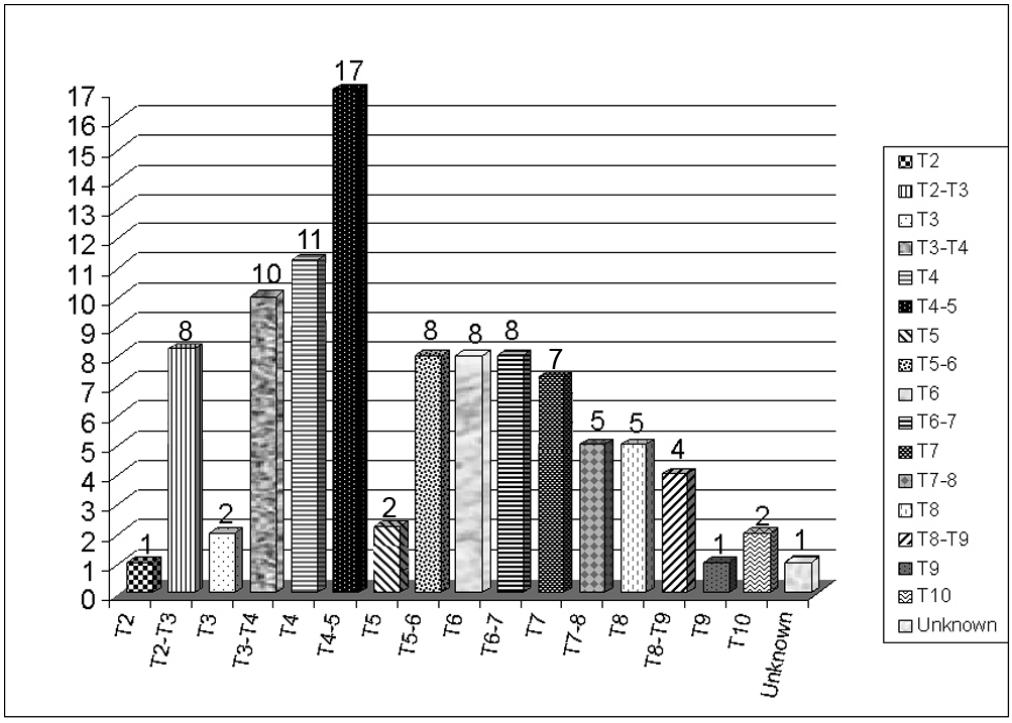

Figure 5. *Frequency of reported idiopathic anterior thoracic spinal cord herniation in the English literature from 1974 to the present.*

funiculus and might cause symptoms of Brown-Séquard syndrome. Review of the literature revealed that 73 (73%) of the 100 reported cases presented with Brown-Séquard syndrome, 19 (19%) with spasticity (mono or paraparesis), and 8 (8%) with numbness or leg pain. Although the dura is sensitive to pain, review of the literature showed that only 48% of the patients had pain (7,9,10,12,13,19,29–32). Sphincter dysfunction, another symptom of ISCH, was reported in 7% of the patients (2,6,17,22). Impotence was a rare symptom of ISCH, reported in only 1 patient (17).

MRI is the gold standard technique for diagnosis of ISCH. On sagittal MRI, typical features are ventral displacement, sharp ventral angulation of thoracic spinal cord, and enlargement of dorsal subarachnoid space (2,16). Phase-contrast MRI can be crucial in excluding a posterior compressing arachnoid cyst and may replace computed tomographic myelography (16,52).

Although some authors selected conservative observant management for patients without motor deficits and no deterioration was noted (2), surgery is still the only method for reversing or stopping the progression of serious neurologic deficits. There are 2 main treatment strategies: (a) closure of the defect by primary sutures or duraplasty after reducing the spinal cord and (b) simply widening the dural defect to prevent strangulation of the cord. The latter technique was first described by Nakazawa (23) and was performed by the authors, who proposed "dural duplication" as the cause of spinal cord herniation. Undoubtedly, this technique is much easier and requires less traction of the spinal cord than repair of the dural defect, but anterior cerebral spinal fluid collection is not an uncommon complication after this procedure. Some authors suggest using a dural patch, even in cases in which there is a clear double dural layer (17). Primary suturing of the dural defect was first performed by Wortzman in 1974 (27). Some authors argued that there is not enough space to pass the needle for primary sutures; therefore, this procedure increases risk of spinal cord damage (33).

A more widely used technique for dural repair is duraplasty. This technique was first described by Masuzawa in 1981 (29). A number of materials were used to obliterate the defect, such as muscle fascial flap, fat, lyophilized dura, and Gortex membrane (17,18,33–35).

In our review, we were able to obtain surgical outcome details for 96 (96%) of the 100 patients; no data were available for 4 patients, so the outcome ratios were based on 96 patients. In summary, 70 (73%) improved, 19 (20%) did not experience any change, and 6 (7%) deteriorated after surgery (Table 2). The outcomes for patients who initially presented with Brown-Séquard syndrome were better than for patients with spastic disorders (paralysis, paraparesis, and monoparesis). Good outcome was reported in 56 of 73 (76.7%) patients with Brown-Séquard syndrome and in 9 of 19 patients (47.3%) with spasticity.

Postoperative T2-hyperintense parenchymal signal in the reduced spinal cord was reported previously and accepted as a predictor of a poor outcome (12,35). We

Table 2. Summary Outcome of Surgical Treatment of Idiopathic Anterior Thoracic Spinal Cord Herniation According to Clinical Manifestation

Presentation	Improved	Unchanged	Deteriorated	Data Not Available	Total
Brown-Séquard	56	12	2	3	73
Spasticity	9	6	3	1	19
Numbness, leg pain	6	1	1	-	8
Total	71	19	6	4	100

postulate that this pathologic signal change may have been caused by vascular impairment during the surgery. In this case, although postoperative MRI revealed increased T2 parenchymal signal and expansion of the cord, the patient experienced improvement in her symptoms.

Based on the literature review, surgery is crucial in the management of this rare entity, and duraplasty is a more widely performed method. Thorough literature review revealed that status improved after surgery for 7 of 8 patients treated by primary suturing of the dural defect. In our opinion, if the reduced spinal cord is not very swollen and the dural defect is longitudinally oriented and narrow, this surgical procedure can be considered for performing blunt dissection of epidural fat planes; moving the table to a 45-degree oblique position might help to avoid excessive cord manipulation.

CONCLUSIONS

In conclusion, ISCH is a rare clinical entity that should be considered in the differential diagnosis of Brown-Séquard syndrome. Review of the literature revealed that the outcome for patients who initially had Brown-Séquard syndrome were significantly better than for patients who initially presented with spastic paralysis. Although progression of neurologic deficits sometimes can be very slow, reduction of the spinal cord and repair of the defect are crucial in stopping or reversing neurologic deterioration.

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